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<th><strong>Title</strong></th>
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<tr>
<td><strong>Authors(s)</strong></td>
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</tr>
<tr>
<td><strong>Publication date</strong></td>
<td>2009-07-01</td>
</tr>
<tr>
<td><strong>Conference details</strong></td>
<td>Computational and Mathematical Biomedical Engineering (CMBE09), Swansea, UK, June 29- July 1, 2009</td>
</tr>
<tr>
<td><strong>Publisher</strong></td>
<td>Computational &amp; Mathematical Biomedical Engineering (CMBE)</td>
</tr>
<tr>
<td><strong>Item record/more information</strong></td>
<td><a href="http://hdl.handle.net/10197/4716">http://hdl.handle.net/10197/4716</a></td>
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ACCURATE PREDICTION OF BLOOD FLOW TRANSIENTS: A FLUID-STRUCTURE INTERACTION APPROACH

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ABSTRACT

Numerical studies are widely employed in establishing blood flow transients in arteries. Unfortunately, many of these are based on rigid arterial geometries where the physiological interaction between the flowing blood and the dynamics of a deforming arterial wall is ignored. Although many recent studies have adopted a fluid-structure interaction (FSI) approach, they lack the necessary validation and, thus, cannot guarantee the accuracy of their predictions. This work employs a well-validated FSI model to establish the dependency of WSS transients on arterial flexibility and predict flow transients in arterial geometries. Results show a high dependency of WSS transients on arterial wall flexibility, with hoop strains of as low as 0.15% showing significant differences in these transients compared to that seen in a rigid geometry. It is also shown that flow in the atherosclerosis susceptible regions of the vascular tree is characterised by a highly disturbed flow. In these regions, WSS magnitudes are at their lowest, while the WSS spatial gradients, rate of change and oscillatory shear index are at their highest.

Key Words: blood flow transients, wall shear stress, fluid-structure interaction.

1 INTRODUCTION

Blood flow through a compliant artery requires appropriate fluid-structure coupling in order to account for the interaction between the flowing blood and the deforming arterial wall. Many numerical models used to predict blood flow are based on rigid arterial geometries [1, 3], where this interaction is not taken into account. Although much attention is given to the complex flow patterns in arteries, the deformation of the arterial wall during each contraction and expansion of the heart is ignored. Some recent studies now use FSI approach to predict blood flow behaviour in arteries [7]. However, many of these models still lack necessary validation and, thus, can not guarantee the accuracy of their predictions.

The current work employs a well-validated FSI model [8] to establish the effect of arterial wall flexibility on WSS transients, thus, the inadequacy of rigid arterial geometries to accurately predict blood flow transients. The model is also used to predict flow transients in a patient-specific carotid artery and, in a related study, to show the effect of local stiffening of the arterial wall due to an emerging atherosclerotic plaque on artery deformation profiles [2]. OpenFOAM, a finite volume method based C++ library [9], was used in this study.

2 NUMERICAL STUDIES

Numerical studies were employed to establish the effect of arterial flexibility on WSS transients and predict of flow transients in the carotid artery bifurcation.
2.1 WSS Transients in Straight Pipes of Varying Stiffness

A parametric study was conducted on flexible pipes of varying stiffness and a rigid pipe in order to establish the effect of pipe wall flexibility and FSI on WSS transients. Different pipe deformations were achieved by varying the pipe stiffness $E$, while the applied axial velocity was kept fixed at 0.76 m/s. This velocity was suddenly applied at the pipe inlet in order to initiate wave propagation along the pipe length. The resulting change in pressure $\Delta p$ due to the applied velocity, $V_x$, is given by $\Delta p = \rho_f C_f V_x$, where $\rho_f$ is the fluid density and $C_f$ is the pressure wave speed [10].

For flexible pipes, $E$ was varied from 4.6 MPa to 120 GPa, resulting in hoop strains of 2.2% to 0.0066%. With $V_x$ kept fixed, it was possible to establish the effect of pipe stiffness on WSS transients. The geometry of the pipe was kept fixed at $d = 10$ mm and $b = 0.5$ mm. The fluid domain was modelled as a compressible Newtonian fluid with dynamic viscosity $\eta = 0.004$ Ns/m$^2$ (representative of the viscosity of blood), density $\rho_f = 998$ kg/m$^3$ and Bulk modulus $K = 2.2$ GPa (which are properties of water at 20°C). Since pipe deformations are relatively small (maximum hoop strain = 2.2%), the solid domain was modelled as a linear elastic solid with density $\rho_s = 1000$ kg/m$^3$ and Poisson ratio $\nu = 0.4995$. More details on problem set-up can be found in [8].

![Figure 1: Variations in WSS transients due to changes in pipe stiffness, E.](image)

Fig. 1 presents WSS transients plotted at 60 mm from the pipe inlet as a function of the position of the propagating pressure wavefront (given by $C_f \times time$). The results clearly show a high dependency of WSS transients on pipe deformations. For a rigid case, there is almost sudden increase in WSS as the wavefront approach the 60 mm position from the inlet, and stays constant thereafter. This is not the case for flexible pipes were these changes are much more gradual. Hoop strains of as low as 0.15% show significant differences in WSS transients compared to that seen in a rigid pipe. Therefore, the flexibility of the arterial wall is an important determinant of WSS transient behaviour.

2.2 Flow transients in a Patient-Specific Carotid Artery

Following the parametric study, the numerical model was employed to predict flow transients in a patient-specific carotid artery. The artery geometry and flow conditions used were based on literature values [6]. The computational mesh was generated in Gambit, using structured multi-block hexahedrals, and then imported into OpenFOAM. Cell density in the vicinity of the bifurcation region is gradually increased in order to achieve a better resolution in this region [8].

A velocity waveform was applied at the carotid inlet while pressure waveforms were specified at its outlets. The flow ratio between the external to internal carotid artery was kept fixed at 40:60 [5], using
a method proposed by [6]. Fluid and solid properties remained the same as in the parametric study except for the Young’s modulus which was now kept fixed at 4.7 MPa, corresponding to the stiffness of mock arteries used in related work [8].

Flow in the artery, especially in the outer edges of the bifurcation, is seen to be characterised by highly disturbed flow (Figures 2(a) and 2(b)). A vortex is also observed to form in this region during the diastole phase (Figure 2(b)). There are also noticeable changes in the position of the vortex over the entire cardiac cycle. These are reported in [8].

![Figure 2: Velocity distribution in the patient specific carotid artery, at different stages of the cardiac cycle: (a) during systolic acceleration and (b) diastole phase.](image)

The highly disturbed flow in the carotid artery bifurcation is likely to contribute to the development of atherosclerotic plaques, which are known to affect the outer edges of the carotid artery bifurcation [4]. Circumferential flow patterns similar to those reported by [5] were also observed, and are reported in [8].

Figures 3(a) and 3(b) shows WSS distribution in the carotid artery, at two different instances of the cardiac cycle, i.e. during systole (t/T_p = 16) and during diastole (t/T_p = 50) phases. WSS is consistently lower along the outer edges of the bifurcation than anywhere else in the artery. The highest WSS spatial gradients and its rate of change also occur in these regions [8].

![Figure 3: Velocity distribution in the patient specific carotid artery, at different stages of the cardiac cycle: (a) cardiac cycle stages, (b) t_1 and (c) t_2.](image)

The predicted oscillatory shear index (OSI), which is a measure of the degree of the oscillation of WSS [8], is presented in Figure 4. Similar to WSS gradients, OSI is highest along the outer edges of the carotid bifurcation with location ”B” showing almost complete flow reversal (≈ 0.5).
3 CONCLUSIONS

WSS transients is highly dependent on arterial flexibility and, thus, rigid arterial geometries can not be used to accurately predict flow transients in arteries. Hoop strains of as low as 0.15% produced significant differences in WSS transients compared to those observed in a rigid geometry. Numerical prediction fo flow transients in the carotid artery bifurcation also showed highly disturbed flow with low WSS and high WSS spatial gradients, WSS temporal gradients and OSI occur in the outer edges of the bifurcation, where atherosclerotic plaques are known to occur [4].

References


